Do race-specific models explain disparities in treatments after acute myocardial infarction?

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Background Racial differences in healthcare are well known, although some have challenged previous research where risk-adjustment assumed covariates affect whites and blacks equally. If incorrect, this assumption may misestimate disparities. We sought to determine whether clinical factors affect treatment decisions for blacks and whites equally.

Methods We used data from the Cardiovascular Cooperative Project for 130709 white and 8286 black patients admitted with an acute myocardial infarction. We examined the rates of receipt of 6 treatments using conventional common-effects models, where covariates affect whites and blacks equally, and race-specific models, where the effect of each covariate can vary by race.

Results The common-effects models showed that blacks were less likely to receive 5 of the 6 treatments (odds ratios 0.64-1.10). The race-specific models displayed nearly identical treatment disparities (odds ratios 0.65-1.07). We found no interaction effect, which systematically suggested the presence of race-specific effects.

Conclusions Race-specific models yield nearly identical estimates of racial disparities to those obtained from conventional models. This suggests that clinical variables, such as hypertension or diabetes, seem to affect treatment decisions equally for whites and blacks. Previously described racial disparities in care are unlikely to be an artifact of misspecified models. (Am Heart J 2007;153:785-91.)

Racial differences in healthcare are widely known; however, the reasons behind these differences are not as well understood. Although clinicians and policy makers worry that these healthcare disparities might reflect provider decisions to treat blacks and whites differently, others question whether the studies are adequately rigorous.[1-3] Critics suggest that inadequate accounting for confounders or inappropriate statistical approaches might overestimate the gaps in care between black and white Americans.[1,4]

Past research focused on accounting for potential confounders, such as patient choice,[5,6] economic differences,[7,8] or differences in access to care[9], but less on the use of appropriate statistical technique. To account for baseline differences between blacks and whites, most studies use multivariate modeling techniques that assume covariates, whose values often differ strikingly, affect blacks and whites equally. For example, in evaluating racial differences in cardiovascular treatments, investigators usually adjust for baseline differences in hypertension rates, assuming that the impact of hypertension on cardiovascular therapy is the same for whites and blacks. However, if physicians weigh the presence of hypertension differently in blacks and whites, simply including this covariate in a multivariable model would be inadequate.

Given the priority and substantial resources dedicated to the issue of racial differences in healthcare, it is critical that the measured racial gap in care not be the partial consequence of inadequate statistical techniques. Specifically, the assumption of "common-effects," that covariates such as hypertension or diabetes affect physician treatment decisions for blacks and whites equally, needs to be tested. If this assumption is not correct, understanding how certain covariates impact physician decisions to treat blacks and whites might offer insights into why racial disparities exist. In addition, the true magnitude of racial disparities in healthcare would need to be carefully reexamined. Alternatively, if the assumption is correct, clinicians and policy makers can dismiss this argument as a potential mechanism for explaining disparities and focus on ensuring equitable care. Therefore, we examined how

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well common-effects models perform compared with other approaches and whether previous findings of disparities would change meaningfully if we used race-specific models that allowed for covariates to have differential effects based on race.

Methods

Data collection

To determine whether racial differences in treatment might be due to race-specific effects of comorbidities, we used data from the Cooperative Cardiovascular Project (CCP), which collected detailed chart-based clinical data on Medicare patients admitted to a hospital for acute myocardial infarction (AMI). The CCP data collection process, detailed elsewhere,[10,11] is briefly described here. The CCP used administrative data to identify patients admitted with an AMI (*International Classification of Diseases, Ninth Revision, Clinical Modification*, principal diagnosis of 410.xx, excluding episodes with a fifth digit of 2, which designates a subsequent episode of care). Among patients with multiple myocardial infarctions (MIs) during the study period, only the first AMI was examined. Our sample consisted of all Medicare beneficiaries admitted during an 8-month period between 1994 and 1995.[11] Detailed clinical data were abstracted from each patient's chart using a standard protocol. For our analysis, we included only whites or blacks and excluded all patients who were transferred from another emergency room or acute care facility.[12]

Variables

For each admission, we categorized patients into 1 of 5 age categories: 65 to 69, 70 to 74, 75 to 79, 80 to 84, and 85 to 99. To be consistent with prior work using these data, we converted the most continuous variables into categorical ones using cutoffs previously chosen.[13] Specifically, we defined hypotension as systolic blood pressure less than 100 mm Hg; renal function, as measured by creatinine levels, into 3 categories (<1.5, 1.5-1.9, or ≥2.0 mg/dL); anemia as hematocrit level less than 30%; low albumin as a level less than 3.0 mg/dL; low ejection fraction as that less than 40%; and high creatine kinase (CK) as a level greater than 1000.[13]

Outcomes

We examined 6 treatments for blacks and whites as our outcomes: the receipt of reperfusion (defined as thrombolysis or percutaneous coronary interventions [PCIs]) within 6 hours, aspirin during the hospitalization, β-blocker during hospitalization, cardiac catheterization within 30 days of admission, PCI within 30 days of admission, and coronary artery bypass graft (CABG) surgery within 30 days.

Statistical analysis

For each of the 6 treatments outlined, we estimated 2 alternative random-effects models to ascertain whether different clinical factors, such as hypertension or diabetes, affect blacks and whites differently. The first model was the conventionally used common-effects model, where we estimate the effect of each covariate on the receipt of treatment for blacks and whites together. Using the models' coefficients and associated standard errors, we adjusted for baseline differences between blacks and whites using age, sex, and each of the covariates available in the CCP that were likely to be associated either with the predictor (race) or one of the outcomes (receiving therapy for AMI). The full list of covariates in our model, presented in Table I, were selected based upon (1) availability within the CCP database, (2) likelihood of influencing physician treatment decisions, and (3) use in prior studies using these data.[10,11,13]

In our second approach, we estimated race-specific models where the effect of each covariate could vary by race. This was accomplished through the inclusion of an additional set of variables, where we included each of the covariates

Table I. Characteristics of patients in the CCP

	Whites, N = 130709, Mean (95% CI)	Blacks, N = 8286, Mean (95% CI)
Demographics		
Age	76.7	75.6
Female sex (%)	48.9	58.8
Admitted from a nursing home (%)	5.8	6.1
Limited mobility (%)	18.7	26.2
Had DNR on file at admission (%)	9.7	6.2
Clinical history		
Previous revascularization (%)	17.5	9.9
Previous myocardial infarction (%)	29.5	29.0
History of CHF (%)	21.5	27.2
Diabetes (%)	29.6	42.1
Hypertension (%)	60.5	79.5
Current tobacco smoker	14.4	17.8
History of a low EF (%)	3.7	4.5
Metastatic cancer (%)	0.8	0.9
History of PVD (%)	10.3	12.6
History of COPD (%)	20.7	17.8
Dementia (%)	6.1	8.3
Clinical features at presentation		
Atrial fibrillation	9.8	6.8
Received CPR (%)	3.4	4.2
Non–Q-wave MI	40.2	42.7
Anterior location of MI	30.7	30.9
Inferior location of MI	19.6	15.9
Other location of MI	9.4	10.5
Heart block (%)	15.9	14.1
Congestive heart failure (%)	28.5	33.2
Hypotension (%)	3.8	3.6
Cardiogenic shock (%)	2.3	2.0
Elevated CK (%)	30.2	33.4
Albumin <3.0 mg/dL (%)	4.4	6.9
Creatinine >2.0 mg/dL (%)	15.4	17.8
Hematocrit <30% (%)	4.5	9.1

All differences were significant at *P* value less than .001, except for the following characteristics: admitted from a nursing home, previous myocardial infarction, history of low ejection fraction, metastatic cancer, anterior location of AMI, hypotension, and cardiogenic shock. *CHF*, Congestive heart failure; *EF*, ejection fraction; *PVD*, peripheral vascular disease; *COPD*, chronic obstructive pulmonary disease; *CPR*, cardiopulmonary resuscitation.

Table II. Rates of treatments (and 95% CI) using race-specific versus common-effect coefficients to adjust* for baseline differences

	Reperfusion within 6 h	Aspirin	β-Blocker	Cardiac catheterization	PCI	CABG within 30 d
Treatments of patients in the CCP						
Actual rate for blacks (%)	11.9 (11.2-12.6)	77.6 (76.7-78.5)	42.4 (41.4-43.5)	39.2 (38.1-40.2)	12.3 (11.6-13.0)	8.9 (8.3-9.5)
Actual rate for whites (%)	18.4 (18.2-18.6)	78.3 (78.1-78.5)	45.6 (45.4-45.9)	46.6 (46.3-46.8)	18.1 (17.9-18.3)	13.7 (13.5-13.9)
Predicted rates using models with common-effects coefficients						
Predicted rate for blacks (%)	13.3 (12.6-14.0)	79.6 (78.7-80.5)	43.4 (42.4-44.4)	42.1 (32.9-35.3)	14.0 (13.3-14.8)	9.2 (8.6-9.8)
Predicted rate for whites (%)	18.3 (18.1-18.5)	78.1 (77.9-78.3)	45.6 (45.3-45.8)	46.4 (46.1-46.6)	17.9 (17.7-18.1)	13.7 (13.5-13.8)
OR†	0.68 (0.65, 0.72)	1.10 (1.03, 1.19)	0.92 (0.88, 0.95)	0.84 (0.81, 0.87)	0.75 (0.70, 0.79)	0.64 (0.59, 0.69)
Predicted rates using models with race-specific coefficients						
Predicted rate for blacks (%)	12.7 (11.8-13.6)	79.3 (78.3-80.3)	42.7 (41.5-44.0)	41.2 (40.2-42.3)	13.8 (12.9-14.7)	9.5 (8.7-10.3)
Predicted rate for whites (%)	18.3 (18.0-0.18.5)	78.1 (77.9-78.3)	45.6 (45.3-45.8)	46.4 (46.1-46.6)	17.9 (17.7-18.1)	13.6 (13.5-13.8)
OR†	0.65 (0.60, 0.70)	1.07 (1.01, 1.14)	0.89 (0.85, 0.94)	0.81 (0.78, 0.85)	0.73 (0.68, 0.79)	0.66 (0.60, 0.73)

*Adjusted for age, sex, source of admission (nursing home, other facility), level of mobility, the presence of a DNR at admission, previous revascularization, prior MI, history of any of the following: CHF, diabetes, hypertension, low ejection fraction, metastatic cancer, peripheral vascular disease, chronic obstructive pulmonary disease, or dementia. Also adjusted for the presence of any of the following at admission: atrial fibrillation, location of MI, heart block, CHF, hypotension, cardiogenic shock, elevated creatine kinase, low albumin, elevated creatinine, or low hematocrit.
†Blacks compared with whites.

interacted with the race indicator variable. Because the common-effects model is nested within the race-specific model (the former lacks the race interactions effects, which are included in the latter), we examined whether predicted values from these 2 models and the resultant odds ratios (OR) on race were substantively different. To formally evaluate whether the 2 models yielded statistically different predictions, we performed a Wald test to determine if the interactions effects were jointly equal to zero.[14] We also computed likelihood-ratio tests to assess the fit of the 2 models and noted that the results were indistinguishable from the conclusion of the Wald tests. The statistical approach to our models is explained in greater detail in the Technical Appendix.

We report bootstrapped standard errors for the predictions based on 100 replications. In all of our random-effects models, we clustered our standard errors at the level of the hospital referral region. The standard errors reported are not sensitive to whether we clustered at this level, the level of a given hospital, or used generalized estimating equations to perform the analysis. We used these standard errors to compute 95% confidence intervals (CIs) for the adjusted rates at which whites and blacks receive each treatment.

All analyses were conducted using STATA 9.0, College Station, TX.

Results

Of the 138995 Medicare beneficiaries hospitalized for AMI in the CCP database, 8286 (6.0%) were blacks. Black Americans were younger, more likely to be female, admitted from a nursing home, have limited mobility, and less likely to have a "do not resuscitate" (DNR) on file at the time of admission (Table I). Blacks had higher rates of hypertension, diabetes, tobacco use, and other comorbidities associated with higher cardiovascular risk. Finally, there were important racial differences in clinical presentation that are outlined further in Table I.

Blacks had lower unadjusted rates of reperfusion within 6 hours of admission and β-blocker use, although the rates of aspirin use were comparable between the 2 groups. By 30 days after admission, black patients had significantly lower rates of cardiac catheterization, PCI, and CABG surgery (Table II).

The common-effects multivariable model demonstrated that blacks were less likely to receive 5 of the 6 therapies (all but aspirin during hospitalization, rows 3 and 4, Table II) with ORs (comparing blacks to whites) that varied from 0.64 (95% CI 0.59-0.69) for CABG within 30 days to 1.10 (1.03-1.19) for aspirin during hospitalization. Our examination of treatment differences using race-specific prediction models revealed nearly identical results (rows 6 and 7, Table II). The predicted rates for whites using race-specific models were nearly identical for each of the 6 outcomes. The predicted rates for blacks using "black-specific" models were also comparable, though generally lower, than using common-effects models (Table II). Finally, the ORs for the common-effects models were nearly identical to those from the race-specific models (rows 5 and 8, Table II), with race-specific models usually demonstrating a slightly larger racial gap in care. Similar results

Table III. Effect of selected patient characteristics on likelihood of receiving 3 selected treatments

	Catheterization		Reperfusion		β-Blocker	
	OR, blacks	OR, whites	OR, blacks	OR, whites	OR, blacks	OR, whites
Female	0.91*	0.75*	0.93	0.93	1.00	0.99
Age						
70-74 y	0.75	0.81	0.94	0.85	0.91	0.96
75-79 y	0.57	0.59	0.82	0.70	0.81	0.89
80-84 y	0.33*	0.27*	0.57	0.48	0.87	0.83
≥85 y	0.09	0.09	0.31	0.28	0.62	0.70
Previous revascularization	1.40	1.24	1.20	0.97	1.04	1.01
Dementia	0.35	0.28	0.30	0.52	0.99	0.89
Metastatic cancer	0.19	0.23	0.62	0.26	0.99	0.97
History of peripheral vascular disease	1.00	0.94	0.86	0.81	1.01	1.07
History of chronic obstructive pulmonary disease	0.84*	0.71*	0.82	0.78	0.55	0.53
History of previous angiogram	1.37	1.32	1.00	1.09	1.62	1.81
Atrial fibrillation at admission	0.73	0.71	0.82	0.82	0.85	0.79
CPR at admission	0.42	0.48	0.55	0.57	0.74	0.72
Anterior MI	0.99	0.94	4.50	4.09	1.04	1.03
Inferior MI	0.97	1.0	4.39	4.79	1.05	1.02
Other MI	0.66	0.69	0.81	0.68	0.64	0.75
Heart block at admission	0.85	0.84	0.74	0.75	0.68	0.78
Hypotension at admission	0.80	0.78	1.46	1.54	0.47	0.50
Elevated CK at admission	1.03	0.98	2.75	2.78	1.25	1.20
Admitted from a nursing home	0.32	0.33	0.56	0.46	0.73	0.72
Admitted from another institution	1.11	0.74	0.92	0.73	1.14	0.95
Unable to walk	0.22*	0.35*	0.37	0.51	0.57	0.69
Walk with assistance	0.58	0.56	0.61	0.61	0.88	0.84
Low albumin at admission	0.94	0.80	1.05	0.98	0.81	0.82
High bilirubin at admission	0.59	0.66	0.48	0.64	0.73	0.88
Low hematocrit at admission	0.65	0.56	0.61	0.56	0.87	0.81
Previous MI	0.83	0.78	1.06*	0.87*	1.12	1.02
History of CHF	0.61	0.59	0.58	0.61	0.62	0.62
History of diabetes mellitus	0.99*	0.81*	1.02*	0.78*	0.93	0.86
History of hypertension	1.18	1.15	1.17*	0.96*	1.46	1.39
History of low ejection fraction	1.02*	0.71*	0.81	0.75	0.90	0.80
History of peripheral vascular disease	1.00	0.94	0.86	0.81	1.01	1.07
Current tobacco smoker	0.94	0.91				
CHF at admission	0.70	0.69	0.63	0.63	0.63	0.62
Shock at admission	1.28	1.12	1.36	1.64	0.52	0.49
DNR at admission	0.27	0.25	0.67	0.71	0.69	0.67
Renal dysfunction						
Creatinine 1.5-2.4 mg/dL	0.85*	0.70*	0.81	0.81	0.87	0.80
Creatinine ≥2.5 mg/dL	0.43	0.37	0.48	0.52	0.70	0.72

Data missing if the models could not create a parameter estimate in a race-specific model. *CPR*, Cardiopulmonary resuscitation; *CHF*, congestive heart failure.
*Represents statistically significant differences at $P < .05$.

were also obtained for the use of angiotensin-converting enzyme inhibitors in the hospital (not reported in the tables). Here, the common-effects model for blacks yielded an adjusted rate of 0.39 (95% CI 0.37-0.40), and the race-specific model yielded an identical adjusted rate of 0.39 (95% CI 0.37-0.40).

Wald and likelihood ratio tests were performed to formally evaluate whether the interaction effects were jointly different from zero. Both tests rejected the null hypothesis that the coefficients on the interaction effects were jointly zero ($P < .001$), but these tests are influenced by the large samples available to us. Even though the 2 models produce estimates that are statistically different (Table II), these differences are small and not of clinical significance. For example, the predicted rate of PCI for blacks in the common-effects model is 14.0% versus 13.8% in the race-specific model.

When we examined the interaction terms, we found them to be inconsistent in size and statistical significance across different treatments. In Table III, we present the results of the interactions between race and patient characteristics for 3 common cardiac treatments. Of the interaction variables presented, only a previous history of diabetes had interactions that were significant ($P < .05$) for 2 of the 3 examined treatments, and 8 other covariates had 1 interaction that was statistically significant (Table III). The ORs for each of these covariates were relatively similar for blacks and whites across all 3 treatments (Table III). We found no covariates where there were important interactions across multiple treatments.

Finally, we examined graphically the relationship between the predicted rate of treatment using common-effect models on the x-axis and the predicted rate using race-specific models on the y-axis. We found that for both groups of patients, the common-effects rates closely predicted the race-specific rates of cardiac catheterization across the entire spectrum of patients, from those who had low predicted rates to those with high predicted rates of treatment (Figure 1, *A*). The results were very similar for β-blocker use (Figure 1, *B*) as well as for the other 4 treatments (data not shown). Because white patients constitute most of the patients, and therefore contribute heavily to the estimation of the common-effects model, it is unsurprising that predictions for these patients are insensitive to the choice of model. However, even for black patients who constitute a small fraction of the overall population, the common-effect models still closely predict the rates found using the race-specific model.

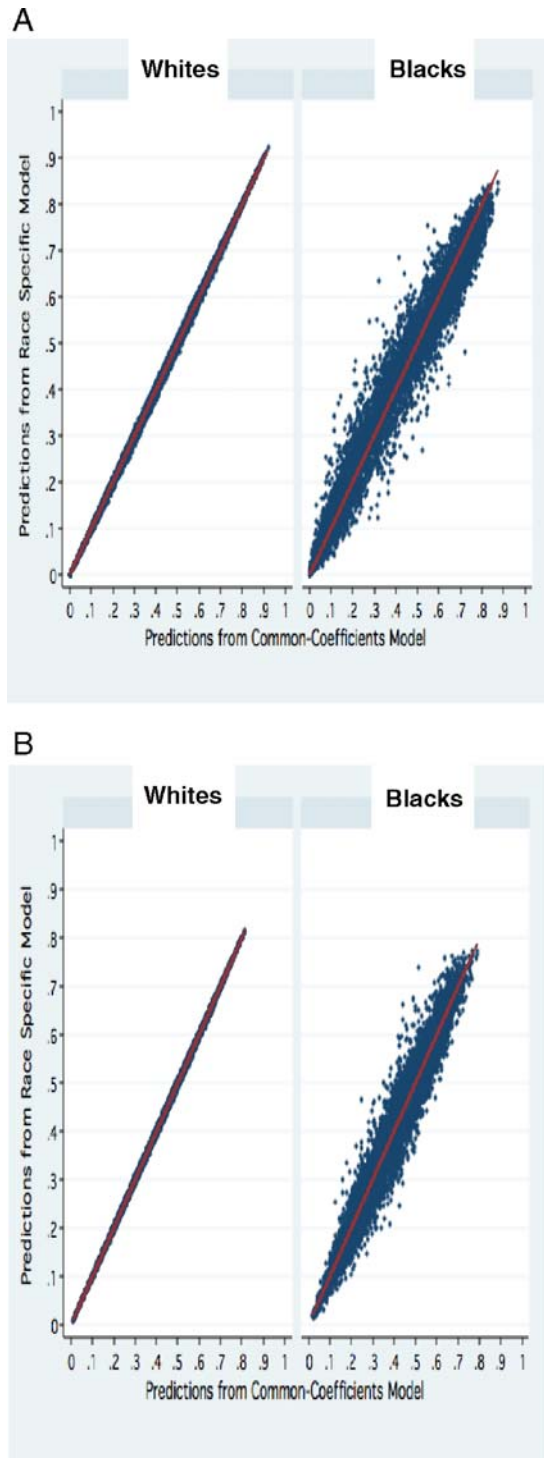
Discussions

We examined whether race-specific coefficients in risk-adjustment models affect the degree of disparities observed for cardiac care and found nearly identical results as common-effects models. For 5 of the 6 outcomes examined, the common-effects models slightly overestimated the rates of treatment for blacks, but only to a small degree. There were no interactions that consistently modified the relationship between race and all of the 6 treatments.

While blacks and whites clearly have different levels of comorbid conditions (ie, diabetes) and important differences in socioeconomic factors, previous research on healthcare disparities has accounted for these differences based on the assumption that clinicians treat comorbidity in the same way for blacks and whites. It is heartening to know that this assumption, whether or not clinically appropriate, has only modest effects on the true relationship between race and treatment outcomes. Although clinicians clearly take these covariates into account in making clinical decisions, we could find no evidence that they weigh these factors differently for whites than they do for blacks. Therefore, our study suggests that an inappropriate common-effects assumption is unlikely to be responsible for treatment disparities seen in cardiac care.

Our results might provide some insight about one potential mechanism for racial differences in treatment: the role of clinician discrimination against minority patients, a factor emphasized by the Institute of Medicine Report as being of paramount importance in explaining racial disparities in healthcare. One might believe that if clinicians discriminated against black patients, they would do so "at the margins." That is, although they might treat white and black patients with clear indications for a treatment the same, among patients who might be marginal candidates for a therapy,

Figure 1



A, Predicted rates of cardiac catheterization using common-effects versus race-specific models for whites and blacks. **B**, Predicted rates of β-blocker use in the hospital from common-effects versus race-specific models for whites and blacks.

blacks may be less likely to be offered the treatment. Under this view, variables such as age and the presence of certain comorbidities (which measure the clinical appropriateness of patients) should affect the receipt of treatment differently in whites and blacks. If this mechanism of discrimination were responsible for disparities in treatment, our race-specific models and our interaction analyses would have likely identified this phenomenon. Our failure to find any consistent interaction effect makes this potential explanation for disparities much less likely. Either physicians discriminate against blacks regardless of clinical appropriateness, or the race effect is proxy for other explanations such as blacks being treated at lower quality facilities.

Our study has important limitations. First, we examined data from the CCP, which are now more than 10 years old, and it is likely that the rates of each of these treatments have risen. However, there is substantial evidence that treatment disparities for patients with AMI have not changed during this time.[15,16] Furthermore, changes in the prevalence of underlying comorbidities would not make our results any less relevant. The main threat to the generalizability of our finding is the unlikely scenario that, over time, the impact of these comorbidities on treatment decisions has changed. Second, our study does not account for contraindications, which is a limitation of the CCP data. We are not aware of any data that demonstrate racial differences in rates of contraindications to these treatments. Therefore, although the ideal rate for all of the therapies is likely less than 100%, it is likely that the ideal rates should not differ substantially by race. Third, we examined treatment differences only and not outcomes. Although the presence of certain comorbidities does not differentially affect the receipt of treatment for whites and blacks, the same may affect downstream survival differently by race. We did not explore this possibility in our analysis primarily because we were interested in clinician decision making and whether physicians weight these covariates differently. Also, given our ability to risk adjust for survival remains limited with data sets such as the CCP, we chose not to examine these outcomes. Finally, we only examined treatment decisions for patients with AMI, and our findings here cannot necessarily be generalized to patients with other conditions.

In conclusion, we examined whether using race-specific models affects the relationship between race and treatment outcomes using a large clinical data set and found minimal effects. The lack of any relationship between comorbidities and differential treatment between whites and blacks suggests that clinicians weigh patient characteristics the same for whites and blacks, at least in cardiovascular care. Therefore, using either race-specific or a more general model is reasonable, and this debate should not distract clinicians and policy makers from the difficult work of understanding and reducing racial disparities in healthcare.

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Appendix A. Technical appendix

To illustrate our technique with a stylized example that incorporates 2 covariates—age and diabetes—for ease of exposition, consider the following equations:

A.1. Common-effects model

This is the conventionally estimated model where the effect of each covariate on the probability of receiving treatment is constrained to be the same by race:

$$Pr(Catheterization = 1) = F(\beta_0 + \beta_1 Black + \beta_2 Age + \beta_3 Diabetes) \quad (1)$$

The function $F(\ )$ indicates the logistic distribution function $F(z) = \exp(z)/[1 + \exp(z)]$. After estimating this model, we calculate the predicted probability of receiving catheterization for each patient as if they were first white, then black. For each observation, we set the race indicator variable on and off to calculate:

$$Pr(Catheterization = 1|White) = F(\beta_0 + \beta_2 Age + \beta_3 Diabetes) \quad (2)$$

$$Pr(Catheterization = 1|Black) = F(\beta_0 + \beta_1 + \beta_2 Age + \beta_3 Diabetes) \quad (3)$$

The (adjusted) racial disparity in the probability of receiving catheterization is the difference between the average probability obtained from Eqs. (2) and (3). This is an adjusted disparity because we used identical distributions of the covariates (age and diabetes) to obtain the predictions for patients as if they were white or black.

A.2. Race-specific model

In this model, age and diabetes can have different effects for whites and blacks on the probability of treatment. Therefore, we estimate the following:

$$Pr(Catheterization = 1) = F(\delta_0 + \delta_1 Black + \delta_2 Age + \delta_3 Diabetes + \delta_4 Black*Age + \delta_5 Black*Diabetes) \quad (4)$$

The variables δ_4 and δ_5 represent the differential effect of age and diabetes on the receipt of treatment for blacks. Statistically, the race-specific model is distinguished from the common-effects model by simultaneously testing $\delta_4 = \delta_5 = 0$ using a Wald or likelihood ratio test. For this reason, the race-specific model is the more general model and nests the common-effects model. To assess whether the different models yield estimates that differ from a clinical perspective, we compare the predictions from each. Predictions from the race-specific model, assuming that all patients are first white and then black, will be given by the following:

$$Pr(Catheterization = 1|White) = F(\delta_0 + \delta_2 Age + \delta_3 Diabetes) \quad (5)$$

$$Pr(Catheterization = 1|Black) = F(\delta_0 + \delta_1 + \delta_2 Age + \delta_3 Diabetes + \delta_4 Age + \delta_5 Diabetes) \quad (6)$$

These predictions will be different than those obtained from the common-effects model as long as δ_4 and δ_5 are different from zero. In contrasting these predictions to ones obtained from the common-effects model, if the treatment δ_4 and δ_5 are positive, using a common-effects approach would overstate racial disparities, and if δ_4 and δ_5 are negative, the common-effects approach would understate disparities. In our tables, we contrast the racial disparity as measured by the difference in the average predictions from Eq. (2) to those in Eq. (5), and those from Eq. (3) to those in Eq. (6). The first comparison provides insights about the degree to which common-effects models produce biased estimates for whites, whereas the second set of comparisons elucidates the degree to which common-effects models produced biased estimates for blacks.

In theory, it is possible that the 2 models yield similar estimates when pooling across all patients, but produce very different estimates for nonstandard patients. For example, it may be the case that the common-effects model produces estimates of receiving catheterization that are considerably different than those from the race-specific model for extremely young or old patients. To examine this possibility, we used each patient's actual values for each covariate and obtained the probability of receiving the treatment from the common-effects and race-specific models. If the 2 models yield similar predictions (not only on average, but throughout the distribution of covariates), then a plot of predictions of one model on those from the other should, on average, align along a 45° line. This provides yet another test of the robustness of the common-effects model vis-à-vis the more general race-specific model by exploiting the full range of covariate values and interactions available in the data.

In other work not reported, we calculated racial disparities from the common-effects and race-specific effects models by predicting the probability of receiving treatment for a patient with the clinical characteristics of the average patient (pooling across whites and blacks), as well as the average black patient and the average white patient. For example, after estimating the common-effects model, we calculated the following:

$$Pr(Catheterization = 1|White) = F\big(\beta_0 + \beta_2 \overline{Age} + \beta_3 \overline{Diabetes}\big)$$

$$Pr(Catheterization = 1|Black) = F\big(\beta_0 + \beta_1 + \beta_2 \overline{Age} + \beta_3 \overline{Diabetes}\big)$$

The bar above each variable denotes explicitly that we evaluated the prediction at the full-sample (combining white and black patients) average value of these covariates. The (adjusted) racial disparity in the probability of receiving catheterization is the difference between the probability obtained from Eqs. (2) and (3). These predictions yielded estimates of the racial disparity that were identical to those reported in this analysis, but produced estimates of the probability of receiving treatment that were substantially different than the observed rates by race. It is only in linear models that the average of the dependent variable is equal to the predicted average at the point of sample means.